no significant difference between the intact side and the ischemic side.

Fig. 5 is a drawing showing the region for measurement of blood vessel area in the non-infarcted ischemic penumbra of the parietal lobe in a 5 μm thick section of the brain. In Fig. 5, the part enclosed with a rectangle is the region for measurement of blood vessel area: cerebrocortical layers II - IV of the parietal lobe; the part painted with black in Fig. 5 is the infarct lesion when ginsenoside Rb_1 was administered; and the part shown in gray in Fig. 5 is the infarcted lesion when physiological saline was administered.

Fig. 6 consists of photographs instead of drawing, which show vascular networks in the non-infarcted ischemic penumbra of the cerebral cortex on the ischemic side, and vascular networks in the corresponding cerebral cortex on the intact side. The upper part shows the intact side and the lower part the ischemic side.

Table 1 shows the ratio of blood vessel area in the non-infarcted ischemic penumbra of the parietal lobe in the brain sections 5 μ m thick. Data are represented by mean ± SE. Statistical analyses are conducted by ANOVA + Fisher's PLSD. N = 5.

Further, paraffin sections prepared from brain samples at the level 3.6 mm posterior to bregma were subjected to Nissl staining, and the ratio of the left thalamic area to the right

thalamic area (ischemic side/intact side × 100) at the same level was measured. The groups administered with ginsenoside Rb_1 showed significantly higher values than the ischemic group administered with vehicle (physiological saline), and they exhibited the values close to that of the sham-operated group.

Results are shown in Table 2.

Table 2

	n	ratio of area (%)	count of nerve cells
physiological saline	8	86.4 ± 8.1	10.1 ± 5.5
Rb_1: 6 μg/day	5	95.9 ± 3.3*	30.6 ± 2.9**
Rb_1: 60 μg/day	8	95.3 ± 2.4*	31.5 ± 3.6**
Sham-operated group	8	98.8 ± 5.3	58.5 ± 4.7

Table 2 shows the ratio of the left thalamic area to the right thalamic area and numbers of nerve cells (neurons) per 0.099 mm^2 of the ventral posterior nucleus of the thalamus. Data are represented as mean ± SE. Statistical analyses were conducted by Mann-Whitney U-test. In Table 2, *: $p < 0.05$, **: $p < 0.01$. Further, in the ventral posterior nucleus of the thalamus (VP thalamic nucleus) having close synaptic connections (fiber connections) with the ischemic core, large numbers of nerve cells significantly survived without suffering from the secondary degeneration in the ischemic groups administered intravenously with ginsenoside Rb_1 as compared with the ischemic group intravenously infused with vehicle (physiological saline).

Example 4 (Effect of intravenous infusion of ginsenoside Rb_1 on rats with spinal cord injuries)

The rats were anesthetized with inhalation of halothane in a mixture of nitrous oxide and oxygen and were loaded with 20 g of compression to the lower thoracic cord for 20 minutes. More than 30 minutes later, ginsenoside Rb_1 dissolved in physiological saline was injected once into the left femoral vein (12 μ g or 60 μ g), thereafter continuous intravenous administration of ginsenoside Rb_1 was performed for 7 days by using an Alza osmotic minipump (12 μg/day or 60 μg/day). Control animals and sham-operated animals were administered with the same amount of physiological saline (vehicle). The open field locomotor scores [Basso, Bettie and Bresnakan (BBB) scores] were measured before loading spinal cord injury, on the day of spinal cord injury and from the 1st day to the 7th day after the spinal cord injury for use as an index of motor functions (Basso D.M. et al., J. Neurotrauma, 13, 343-359, 1996).

Fig. 8A shows the result of a control rat administered with physiological saline on the 2nd day after spinal cord injury, and Fig. 8B shows the result of a rat administered with ginsenoside Rb_1 (60 μg/day) on the 2nd day after spinal cord injury. As shown in Fig. 8A, the rat, to which physiological saline was administered and the compression of 20 g was loaded on the lower thoracic cord for 20 minutes, obviously exhibited